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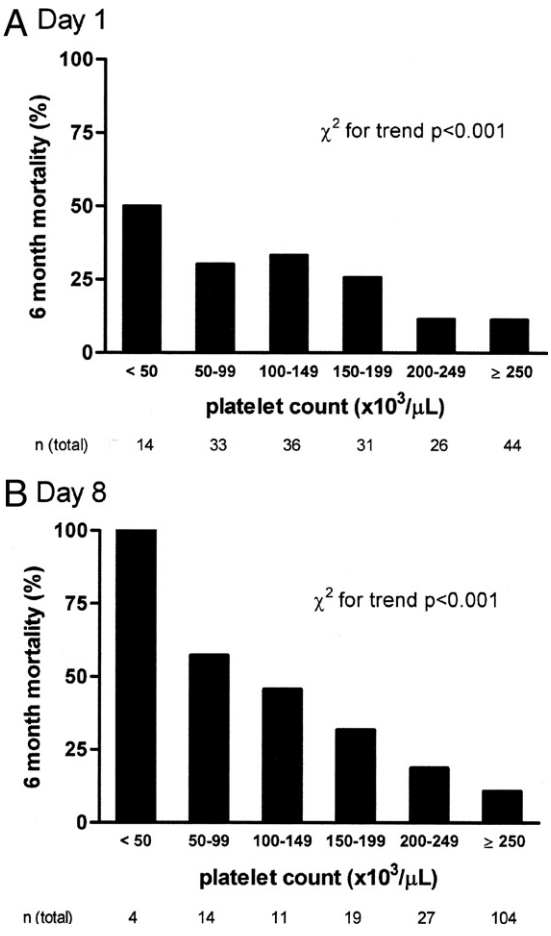

Figure 1 **Severity of Thrombocytopenia Is Associated With 6-Month Mortality**

Six-month mortality in relation to platelet count at day 1 **(A)** and at day 8 **(B)**. The total number of patients within each band of platelet counts is indicated by n. Chi-square test for trend $p < 0.001$ at day 1 and day 8.

not independently predictive on multivariate analysis. In addition, there was no association between erythrocyte sedimentation rate and mortality.

The finding of thrombocytopenia in patients with endocarditis has clinical implications. First, patients presenting with thrombocytopenia should receive empirical antistaphylococcal therapy because of the strong association between baseline thrombocytopenia and *Staphylococcus aureus* infection. Second, if antiplatelet agents are being considered as adjunctive therapy (4), clinicians should exclude coexistent thrombocytopenia because of its potential to increase the risk of bleeding. Third, thrombocytopenia at day 8 indicates an impaired host response to sepsis and predicts increased mortality. In this setting, patients with thrombocytopenia may warrant more intensive monitoring, alterations to treatment, and, where relevant, consideration of surgery.

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Raymond W. Sy, MBBS
Chirapan Chawantanpipat, MBBS
David R. Richmond, MBChB, MSc
***Leonard Kritharides, MBBS, PhD**

*Department of Cardiology
3rd Floor West
Concord Repatriation General Hospital
Hospital Road, Concord
New South Wales
Australia, 2139
E-mail: lenk@med.usyd.edu.au

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APPENDIX

For a supplementary figure and tables, please see the online version of this paper.

Letters to the Editor

Perioperative Myocardial Infarction Has Been Forgotten

Thygesen et al. (1) have published a consensus report that reviews the definition of myocardial infarction (MI) and proposes a new classification of 5 categories based on differences in pathophysiology. We believe that this definition is flawed in 1 respect: it does not mention perioperative MI. This is not the first time that a MI definition has been questioned. When the consensus document of the Joint European Society of Cardiology/American College of Cardiology redefinition of MI was released, Tunstall-Pedoe (2)

indicated several problems with that document. Some of those problems have been solved in this new classification (e.g., the inclusion of fatal cases of MI). This new classification includes spontaneous MI (type 1), MI secondary to ischemia due to either increased oxygen demand or reduced supply (type 2), sudden cardiac deaths or cardiac arrest (type 3), MI associated with percutaneous coronary intervention (type 4), and MI associated with coronary artery bypass grafting (type 5). This classification could be useful to develop future studies analyzing different treatments according to the group to which the patient belongs. However, it is our opinion that an important group of patients has been forgotten: those with MI related to noncardiac surgeries. The etiology and pathophysiology of myocardial ischemia and infarction in this setting are still controversial subjects and could fit either in types 1 or 2. Based on pathology studies (3,4), we believe that perioperative MI have similar pathophysiology to spontaneous MI; therefore, they should be treated the same way. As a complement of Thygesen's classification, we suggest the inclusion of MI after noncardiac surgeries in type 1 MI of the new classification because this inclusion may have implications for the management of acute coronary syndromes in this setting.

***Danielle Menosi Gualandro, MD**
Bruno Caramelli, MD, PhD
Pai Ching Yu, MD
Andre Coelho Marques, MD
Daniela Calderaro, MD

*Heart Institute
University of São Paulo Medical School
Av. Dr. Eneas de Carvalho Aguiar
44 Cerqueira Cesar
São Paulo—SP, CEP 05403000
Brazil
E-mail: danielle.gualandro@incor.usp.br

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Reply

We thank Dr. Gualandro and colleagues for their thoughtful letter. We have considered a number of different clinical scenarios but decided not to target every specific clinical situation, because there are too many to be contained within the framework of the European Society of Cardiology/American College of Cardiology Foundation/American Heart Association/World Heart Federation expert consensus document (1).

We agree that there is a great deal to learn about perioperative myocardial infarctions as the pathophysiology of these differs somewhat from that of myocardial infarction occurring in the usual setting. We also agree that it can be hard to tell whether these infarctions are type 1 or type 2. However, there are some data to guide us.

Studies of patients undergoing noncardiac surgery strongly support the concept that many of the infarctions diagnosed in this connection are caused by prolonged imbalance between myocardial oxygen supply and demand on the background of coronary artery disease (2,3), which together with rise and fall of cardiac markers points toward myocardial infarction type 2.

The fact that many such patients have type 2 infarctions should not obscure the likelihood that some of the infarctions are type 1 as well. Pathology of fatal peri- or post-operative myocardial infarctions shows plaque rupture and platelet aggregation leading to thrombus formation in approximately half of these events (4). Given the differences that likely exist in the therapeutic approaches to each, close clinical scrutiny to identify this group is essential.

Some patients may not have myocardial infarction at all. Careful clinical evaluation including a detailed history, examination, and evaluation of further investigations to identify and treat those with pulmonary embolism, sepsis, and/or the many other conditions associated with myocyte necrosis and troponin elevations is strongly advocated (1).

Although we cannot make criteria for all clinical judgments such as this one, the available information suggests that the use of contemporary troponin assays (5,6) and the decision levels advocated by the expert consensus document (1) maximizes the ability to identify patients with this diagnosis and then to configure the care according to the type based on that judgment.

***Kristian Thygesen, MD, DMSc**
Joseph S. Alpert, MD
Allan S. Jaffe, MD
Harvey D. White, MD, DSc
on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction

*Department of Medicine and Cardiology A
Aarhus University Hospital
Tage-Hansens Gade 2
DK-8000 Aarhus C
Denmark
E-mail: Kristian.Thygesen@as.aaa.dk

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